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=> dup rem 13

PROCESSING COMPLETED FOR L3

L4 9 DUP REM L3 (13 DUPLICATES REMOVED)

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S COWSER, IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system. For a list of commands available to you in the current file, enter "HELP COMMANDS" at an arrow prompt (=>).

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=> s cowsert, L?/au L8 438 COWSERT, L?/AU

=> s 15 or 18 L9 1233 L5 OR L8

=> d his'

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In a multifile environment, a format can only be used if it is valid in at least one of the files. Refer to file specific help messages

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Entered Medline: 20030122

AB The study of signal transduction processes using antisense oligonucleotides is often complicated by low intracellular stability of the antisense reagents or by nonspecific effects that cause

toxicity. Here, we introduce a new class of antisense molecules, so-called GeneBlocs, which are characterized by improved stability, high target RNA specificity, and low toxicity. GeneBlocs allow for efficient downregulation of mRNA expression at nanomolar concentrations, and they do not interfere with cell proliferation. We demonstrate these beneficial properties using a positive readout system. GeneBloc-mediated inhibition of tumor suppressor PTEN (phosphatase and tension homologue detected on chromosome 10) expression leads to hyperactivation of the phosphatidylinositol (PI) 3-kinase pathway, thereby mimicking the loss of PTEN function and its early consequences observed in mammalian cancer cells. Specifically, cells treated with PTEN GeneBlocs show functional activation of Akt, a downstream effector of PI 3-kinase signaling, and exhibit enhanced proliferation when seeded on a basement membrane matrix. In addition, GeneBlocs targeting the catalytic subunit of PI 3-kinase, p110, specifically inhibit signal transduction of endogenous or recombinant PI 3-kinase. This demonstrates that GeneBlocs are powerful tools to analyze and to modulate signal transduction processes and, therefore, represent alternative reagents for the validation of gene function.

## => d 112 ibib abs 2-9

L12 ANSWER 2 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

DUPLICATE 1

2001:253065 BIOSIS ACCESSION NUMBER:

DOCUMENT NUMBER: PREV200100253065

TITLE: Antisense modulation of PI3

kinase p110 beta expression.
Monia, Brett P. [Inventor]; Cowsert, Lex AUTHOR(S):

M. [Inventor]

CORPORATE SOURCE: ASSIGNEE: Isis Pharmaceutical Inc.

PATENT INFORMATION: US 6133032 October 17, 2000

SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct. 17, 2000) Vol. 1239, No. 3. e-file.

CODEN: OGUPE7. ISSN: 0098-1133.

DOCUMENT TYPE: Patent

LANGUAGE: English

ENTRY DATE: Entered STN: 23 May 2001

Last Updated on STN: 19 Feb 2002

AΒ Antisense compounds, compositions and methods are provided for

modulating the expression of PI3 kinase p110

beta. The compositions comprise antisense compounds,

particularly antisense oligonucleotides, targeted to nucleic

acids encoding PI3 kinase p110 beta.

Methods of using these compounds for modulation of PI3

kinase p110 beta expression and for treatment of diseases associated with expression of PI3 kinase

p110 beta are provided.

L12 ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

DUPLICATE 2

2000:447933 BIOSIS ACCESSION NUMBER: DOCUMENT NUMBER: PREV200000447933

TITLE: Antisense modulation of PI3

kinase p110 delta expression.

AUTHOR(S): Monia, Brett P. [Inventor]; Cowsert, Lex

M. [Inventor]

ASSIGNEE: Isis Pharmaceuticals Inc. CORPORATE SOURCE:

PATENT INFORMATION: US 6046049 April 04, 2000

SOURCE: Official Gazette of the United States Patent and Trademark

Office Patents, (Apr. 4, 2000) Vol. 1233, No. 1. e-file.

CODEN: OGUPE7. ISSN: 0098-1133.

DOCUMENT TYPE:

Patent

LANGUAGE:

English

ENTRY DATE:

Entered STN: 18 Oct 2000

Last Updated on STN: 10 Jan 2002

AB Antisense compounds, compositions and methods are provided for

modulating the expression of PI3 kinase p110

delta. The compositions comprise antisense compounds,

particularly antisense oligonucleotides, targeted to nucleic`

acids encoding PI3 kinase p110 delta.

Methods of using these compounds for modulation of PI3

kinase p110 delta expression and for treatment of
diseases associated with expression of PI3 kinase

pl10 delta are provided.

L12 ANSWER 4 OF 9

MEDLINE on STN

ACCESSION NUMBER:

2000482758 MEDLINE

DOCUMENT NUMBER:

PubMed ID: 10850853

TITLE:

IRAK-2 and PI 3-kinase synergistically activate NF-kappaB

and AP-1.

AUTHOR:

Guo F; Wu S

CORPORATE SOURCE:

Institute of Pharmaceutic Sciences, The First Military

Medical University, Guangzhou, China.

SOURCE:

Inflammation, (2000 Aug) 24 (4) 305-16. Journal code: 7600105. ISSN: 0360-3997.

PUB. COUNTRY:

United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200010

ENTRY DATE:

Entered STN: 20001019

Last Updated on STN: 20001019

Entered Medline: 20001012

AB Antisense interleukin-1 (IL-1) receptor associated kinase-2

(IRAK-2) oligonucleotide (ODN) and antisense p110

PI 3-kinase ODN blocked IRAK-2 and

p110 PI 3-kinase expression,

respectively. As a result, antisense IRAK-2 ODN or

antisense p110 PI 3-kinase

ODN inhibited IL-1-induced NF-kappaB and AP-1 activation in HepG2 cells.

The inhibition of NF-kappaB activation by antisense IRAK-2 ODN

or antisense p110 PI 3-

kinase ODN and the inhibition of AP-1 activation by

antisense IRAK-2 ODN were incomplete, whereas AP-1 activation

could be inhibited by antisense p110 PI

3-kinase ODN completely. These results indicate that

IRAK-2 is necessary but insufficient to activate NF-kappaB and AP-1 completely and that although PI 3-kinase is not sufficient for NF-kappaB full activation, it is sufficient to activate AP-1 completely. The effects of IRAK-2 or PI 3-kinase on NF-kappaB and AP-1 activation were confirmed by the results that overexpression of IRAK-2 failed to fully

activate NF-kappaB and AP-1 and that overexpression of p110

PI 3-kinase is insufficient for NF-kappaB full

activation but sufficient for AP-1 activation. Cotransfection experiments showed that the combination of antisense IRAK-2 ODN and

antisense pl10 PI 3-kinase

ODN resulted in additive inhibition of NF-kappaB as well as AP-1

activation. On the other hand, coexpression of IRAK-2 with p110

PI 3-kinase led to a synergistic activation of

NF-kappaB and AP-1. These data suggest that IRAK-2 and PI 3-kinase cooperate to activate NF-kappaB and AP-1.

L12 ANSWER 5 OF 9 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:

130:151352 CA

TITLE:

Distinct roles for the  $p110\alpha$  and hVPS34

phosphatidylinositol 3'-kinases in vesicular

trafficking, regulation of the actin cytoskeleton, and

mitogenesis

AUTHOR (S):

Siddhanta, Uma; McIlroy, James; Shah, Amishi; Zhang,

Yitao; Backer, Jonathan M.

CORPORATE SOURCE:

Department of Molecular Pharmacology, Albert Einstein

College of Medicine, Bronx, NY, 10461, USA

SOURCE:

Journal of Cell Biology (1998), 143(6), 1647-1659

CODEN: JCLBA3; ISSN: 0021-9525

PUBLISHER:

Rockefeller University Press

DOCUMENT TYPE:

Journal

LANGUAGE:

English

The authors have examined the roles of the  $p85/p110\alpha$  and hVPS34phosphatidylinositol (PI) 3'-kinases in cellular signaling using inhibitory isoform-specific antibodies. They raised anti-hVPS34 and anti-pl10 $\alpha$  antibodies that specifically inhibit recombinant hVPS34 and  $pl10\alpha$ , resp., in vitro. They used the antibodies to study cellular processes that are sensitive to low-dose wortmannin. The antibodies had distinct effects on the actin cytoskeleton; microinjection of anti-p110 $\alpha$  antibodies blocked insulin-stimulated ruffling, whereas anti-hVPS34 antibodies had no effect. The antibodies also had different effects on vesicular trafficking. Microinjection of inhibitory anti-hVPS34 antibodies, but not anti-p110 $\alpha$  antibodies, blocked the transit of internalized PDGF receptors to a perinuclear compartment, and disrupted the localization of the early endosomal protein EEA1. Microinjection of anti-pl10 $\alpha$  antibodies, and to a lesser extent anti-hVPS34 antibodies, reduced the rate of transferrin recycling in CHO cells. Surprisingly, both antibodies inhibited insulin-stimulated DNA synthesis by 80%. Injection of cells with antisense

oligonucleotides derived from the hVPS34 sequence also blocked insulin-stimulated DNA synthesis, whereas scrambled oligonucleotides had no effect. Interestingly, the requirement for  $p110\alpha$  and hVPS34occurred at different times during the G1-S transition. These r data suggest that different PI 3'-kinases play distinct regulatory roles in the cell, and document an unexpected role for hVPS34 during insulin-stimulated mitogenesis.

REFERENCE COUNT:

THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS 64 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 9 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:

TITLE:

INVENTOR(S):

SOURCE:

128:58975 CA

Human phosphatidylinositol 3-kinase and its cloning, characterization, and enhanced expression in melanomas

Vanhasebroeck, Bart; Waterfield, Michael Derek Ludwig Institute for Cancer Research, Switz.; Vanhasebroeck, Bart; Waterfield, Michael Derek

PCT Int. Appl., 72 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent English

LANGUAGE:

PATENT ASSIGNEE(S):

FAMILY ACC. NUM. COUNT: PATENT INFORMATION:

> PATENT NO. KIND DATE APPLICATION NO. DATE \_\_\_\_\_\_ \_\_\_\_\_ 19971211 WO 1997-GB1471 19970530 WO 9746688 A1 W: AU, CA, CN, JP, KR, NZ, US RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE AA 19971211 CA 1997-2256483 19970530 CA 2256483 AU 9729705 A1 19980105 AU 1997-29705 19970530 AU 719354 B2 20000504 19970530 19990512 EP 1997-924137 EP 914448 A1

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI CN 1997-195151 19970530 CN 1220701 Α 19990623 NZ 1997-332634 19970530 20000929 NZ 332634 Α JP 1998-500305 19970530 20001226 JP 2000517165 T2 US 1998-194640 19981201 20021119 В1 US 6482623 US 2002-162160 20020603 Α1 20030529 US 2003099627 GB 1996-11460 A 19960601 PRIORITY APPLN. INFO.: W 19970530 WO 1997-GB1471 A3 19981201 US 1998-194640

AB The invention relates to a novel lipid kinase termed pl10 $\delta$  which is part of the phosphatidylinositol 3-kinase (PI3) Kinase family. Human

PI3 Kinase p110 $\delta$  interacts with p85, has

a broad phosphoinositide specificity, and is sensitive to the same kinase inhibitors as PI3 Kinase pl10  $\alpha.\,$ 

However in contrast to previously identified PI3 Kinases which show a ubiquitous pattern of expression, pl10 $\delta$  is selectively expressed in leukocytes. Importantly, pl10 $\delta$  shows enhanced expression in most melanomas tested and therefore may play a crucial role in regulating the metastatic property exhibited by melanomas. Further, pl10 $\delta$  undergoes autophosphorylation in a Mn2+-dependent manner, which hinders the lipid kinase activity of the protein. The identification of agents that enhance or reduce pl10 $\delta$  activity may therefore prevent cancer metastasis.

L12 ANSWER 7 OF 9 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:

125:140241 CA

TITLE:

Association of phosphatidylinositol 3 kinase to

protein kinase C ζ during interleukin-2

stimulation

AUTHOR(S):

Gomez, Javier; Martinez-A., Carlos; Garcia, Alphonse;

Rebollo, Angelita

CORPORATE SOURCE:

Centro Nacional Biotecnologia, Universidad Autonoma,

Madrid, E-28049, Spain

SOURCE:

European Journal of Immunology (1996), 26(8),

1781-1787

CODEN: EJIMAF; ISSN: 0014-2980

PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English

Interleukin-2 induces a serine-phosphorylated phosphatidylinositol 3 kinase activity in the mouse T cell line  $TS1\alpha\beta$ . Moreover, protein kinase C (PKC)  $\zeta$  directly or indirectly assocs. with the phosphatidylinositol 3 kinase and the association appears to be necessary for the serine-phosphorylated phosphatidylinositol 3 kinase activity, since release of  $\zeta$ PKC by competition of binding with peptides spanning the p110 sequence from amino acids 907 to 925 abolishes the serine-phosphorylated phosphatidylinositol 3 kinase activity. This kinase activity is also blocked when  $\zeta$ PKC expression is inhibited by antisense oligonucleotide. Inhibition of phosphatidylinositol 3 kinase activity by wortmannin does not abolish  $\zeta$ PKC association

L12 ANSWER 8 OF 9 MEDLINE ON STN ACCESSION NUMBER: 96009592 MEDLINE DOCUMENT NUMBER: PubMed ID: 7565716

TITLE:

A phosphatidylinositol (PI) kinase gene family in Dictyostelium discoideum: biological roles of putative

mammalian p110 and yeast Vps34p PI 3-kinase homologs during

growth and development.

AUTHOR:

Zhou K; Takegawa K; Emr S D; Firtel R A

CORPORATE SOURCE: Department of Biology, Howard Hughes Medical Institute,

University of California, San Diego, La Jolla 92093-0634,

USA.

CONTRACT NUMBER:

CA60559 (NCI)

SOURCE:

Molecular and cellular biology, (1995 Oct) 15 (10) 5645-56.

Journal code: 8109087. ISSN: 0270-7306.

PUB. COUNTRY:

United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

OTHER SOURCE:

GENBANK-U23476; GENBANK-U23477; GENBANK-U23478;

GENBANK-U23479; GENBANK-U23480

ENTRY MONTH:

199510

ENTRY DATE:

Entered STN: 19951227

Last Updated on STN: 20021218 Entered Medline: 19951025

Three groups of phosphatidylinositol (PI) kinases convert PI into PI(3) phosphate, PI(4) phosphate, PI(4,5) bisphosphate, and PI(3,4,5) trisphosphate. These phosphoinositides have been shown to function in vesicle-mediated protein sorting, and they serve as second-messenger signaling molecules for regulating cell growth. To further elucidate the mechanism of regulation and function of phosphoinositides, we cloned genes encoding five putative PI kinases from Dictyostelium discoideum. Database analysis indicates that D. discoideum PIKI (DdPIKI), -2, and -3 are most closely related to the mammalian

p110 PI 3-kinase, DdPIK5 is closest to the yeast Vps34p PI 3-kinase, and DdPIK4 is most homologous to PI 4-kinases. Together with other known PI kinases, a superfamily of PI kinase genes has been defined, with all of the encoded proteins sharing a common highly conserved catalytic core domain. DdPIK1, -2, and -3 may have redundant functions because disruption of any single gene had no effect on D. discoideum growth or development. However, strains in which both of the two most highly related genes, DdPIK1 and DdPIK2, were disrupted showed both growth and developmental defects, while double knockouts of DdPIK1 and DdPIK3 and DdPIK2 and DdPIK3 appear to be lethal. The delta Ddpik1 delta Ddpik2 null cells were smaller than wild-type cells and grew slowly both in association with bacteria and in axenic medium when attached to petri plates but were unable to grow in suspension in axenic medium. When delta Ddpik1 delta Ddpik2 null cells were plated for multicellular development, they formed aggregates having multiple tips and produced abnormal fruiting bodies. Antisense expression of DdPIK5 (a putative homolog of the Saccharomyces cerevisiae VPS34) led to a defect in the growth of D. discoideum cells on bacterial lawns and abnormal development. DdPIK5 complemented the temperature-sensitive growth defect of a Schizosaccharomyces pombe delta Svps34 mutant strain, suggesting DdPIK5 encodes a functional homolog of yeast Vps34p. These observations indicate that in D. discoideum, different PI kinases regulate distinct cellular processes, including cell growth, development, and protein trafficking.

L12 ANSWER 9 OF 9 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:

120:100544 CA

TITLE:

Cloning and expression of a cDNA for a subunit of PI3

kinase

INVENTOR(S):

Hiles, Ian D.; Fry, Michael J.; Dhand, Ritu; Waterfield, Michael D.; Parker, Peter J.; Otsu, Masayuki; Panayotou, George; Volinia, Stefano; Gout,

Ivan

PATENT ASSIGNEE(S):

Ludwig Institute for Cancer Research, Barbados

SOURCE: PCT Int. Appl., 146 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent English

LANGUAGE:

: 1

FAMILY ACC. NUM. COUNT: PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9321328 W: AU, CA, JP,		19931028	WO 1993-GB761	,	19930413
RW: AT, BE, CH,	DE, DK	, ES, FR,	GB, GR, IE, IT, LU,	MC, 1	NL, PT, SE
AU 9339017	A1	19931118	AU 1993-39017		19930413
AU 664893	B2	19951207			
EP 590126			EP 1993-908028		
R: AT, BE, CH,	DE, DK	, ES, FR,	GB, GR, IE, IT, LI,	LU, 1	MC, NL, PT, SE
JP 06510207		19941117	JP 1993-518112		19930413
JP 07203963	A2	19950808	JP 1994-4313		19940120
JP 3471879	B2	20031202			
US 5824492	A.	19981020	US 1994-162081		19940207
US 5846824	A	19981208	US 1997-780872		19970109
US 6274327	B1	20010814	US 1998-85957		
PRIORITY APPLN. INFO.:			GB 1992-8135	Α	19920413
			WO 1993-GB761	Α	19930413
			US 1994-162081	A.	3 19940207
			US 1997-780872	. A.	3 19970109

PI3 kinase activity is manufactured by expression of the cloned genes in insect cell culture. Affinity purification of the protein from bovine brain using Y751 phosphopeptide from PDGF- $\beta$  receptor as the affinity ligand identified the p85 and p110 (actual mol. weight 124 kDa) proteins that bound to the column with very high affinity and appeared to form a complex. A cDNA bank from SGBAF-1 cells in  $\lambda \text{Uni-ZAP}$  was screened with amino acid sequence-derived oligonucleotide probes from the p110 protein. and the cDNA expressed in Sf9 cells using a baculovirus vector. A cDNA clone was expressed in Sf9 cells using a baculovirus vector to give a detectable PI3 kinase activity. Tissue specificity of gene expression is determined and the corresponding human cDNA was cloned.

C12N015/00.

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## Search Results - Record(s) 1 through 10 of 10 returned.

Scarci results - record(s) 1 unough 10 of 10 feather.
1. <u>US 6046049A</u> . New <u>antisense</u> compounds targeting nucleic acids encoding human <u>PI3 kinase p110</u> delta useful for treating a disease or condition associated with <u>PI3 kinase p110</u> delta expression, e.g. rheumatoid arthritis, asthma. COWSERT, L M, et al. A61K031/7125 A61K048/00 A61P011/06 A61P019/02 A61P029/00 A61P043/00 C07H021/04 C12N015/00 C12N015/09 C12N015/11 C12Q001/68.
2. <u>6046049</u> . 19 Jul 99; 04 Apr 00. <u>Antisense</u> modulation of <u>PI3 kinase p110</u> delta expression. Monia; Brett P., et al. 435/375; 435/366 435/6 435/91.1 536/23.1 536/24.31 536/24.33 536/24.5. C07H021/04 C12Q001/68 C12N015/00.
3. <u>5846824</u> . 09 Jan 97; 08 Dec 98. Polypeptides having kinase activity, their preparation and use. Hiles; Ian D., et al. 435/348; 435/320.1 435/325 536/23.2 536/24.3. C12N005/10 C12N005/16 C12N015/54 C12N015/63.
4. <u>6274327</u> . 27 May 98; 14 Aug 01. Polypeptides having kinase activity, their preparation and use. Hiles; Ian D., et al. 435/7.1; 435/15 435/194 435/21 435/252.3 435/348 435/6 435/69.1 435/69.2 435/7.2 435/7.8 530/350 530/388.26. G01N033/53 C12Q001/68 C12Q001/48 C12Q001/42.
5. <u>5824492</u> . 07 Feb 94; 20 Oct 98. Polypeptides having kinase activity, their preparation and use. Hiles; Ian D., et al. 435/15; 435/194 435/29. C12N009/12 C12Q001/48.
6. 20010016332. 07 Aug 97. 23 Aug 01. AGE-1 POLYPEPTIDES AND RELATED MOLECULES AND METHODS. RUVKUN, GARY, et al. 435/69.1; 424/9.1 435/320.1 435/325 435/375 800/21 800/3 800/8 C12N015/63 A61K049/00 C12N005/00.
7. <u>5650293</u> . 10 Jun 94; 22 Jul 97. Nucleic acid encoding pp60.sup.PIK and the methods of making pp60.sup.PIK. White; Morris F 435/69.1; 435/252.3 435/320.1 435/354 536/23.5. C12P021/06 C12N005/00 C12N001/20 C07H021/04.
8. <u>5985589</u> . 06 Jan 99; 16 Nov 99. Lipid kinase. Chantry; David H., et al. 435/15; 435/194 435/252.3 435/320.1 435/69.1 435/69.2 435/7.7 530/350 536/23.2 536/23.5. C12Q001/48 C12N009/12 C12P021/06 C07H021/04.
9. <u>5882910</u> . 25 Nov 97; 16 Mar 99. Lipid kinase. Chantry; David H., et al. 435/194; 435/252.3 435/320.1 435/69.1 435/69.2 435/7.7 530/350 536/23.2 536/23.5. C12N009/12 C12N001/20 C12P021/06 C07H021/04.
10. <u>6482623</u> . 01 Dec 98; 19 Nov 02. Lipid kinase. Vanhaesebroeck; Bart, et al. 435/194; 435/183 435/252 3 435/320 1 435/6 536/23 2. C12N009/12 C12N021/06 C12N009/00 C12N001/20

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L5 and (@ad<19990720)	10		

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## **Search Results -** Record(s) 1 through 8 of 8 returned.

1. <u>US 6046049A</u> . New <u>antisense</u> compounds targeting nucleic acids encoding human <u>PI3 kinase p110</u> delta useful for treating a disease or condition associated with <u>PI3 kinase p110</u> delta expression, e.g. rheumatoid arthritis, asthma. COWSERT, L M, et al. A61K031/7125 A61K048/00 A61P011/06 A61P019/02 A61P029/00 A61P043/00 C07H021/04 C12N015/00 C12N015/09 C12N015/11 C12Q001/68.
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